Evaluation of Seedling Resistance to Tan Spot and Stagonospora nodorum Blotch in Tetraploid Wheat

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ABSTRACT

Tetraploid durum wheat (*Triticum turgidum* L. subsp. *durum*), an important cereal used for making pasta products, is more vulnerable to various wheat diseases than bread wheat (*T. aestivum* L.). To identify resistant sources useful for improving durum resistance to tan spot [caused by *Pyrenophora tritici-repentis* (Died.) Drechs.] and Stagonospora nodorum blotch (SNB) [caused by *Phaeosphaeria nodorum* (E. Müller) Hedjaroude], we evaluated 688 accessions belonging to *T. turgidum* L. subspecies *T. carthlicum*, *T. polonicum*, *T. turgidum*, *T. dicoccum*, and *T. turanicum* for their seedling resistance to *P. tritici-repentis* and *P. nodorum*. Accessions were inoculated with a *P. tritici-repentis* race 1 isolate (Pti2) and a mixture of three diverse isolates of *P. nodorum* (LDNSn4, BBCSn5, and Sn2000). Then 206 accessions with low and intermediate disease reaction to either of the inocula were further evaluated for reactions to *P. tritici-repentis* and *P. nodorum* and for sensitivity to host-selective toxins produced by the two pathogens. Data showed that 25 and 132 accessions had high levels of or partial resistance to tan spot and SNB, respectively, with 10 accessions, including *T. dicoccum* and *T. turgidum,* showing resistance to both diseases. The resistant accessions identified in this study would be particularly useful for developing durum wheat germplasm resistant to tan spot and SNB due to their semidomesticated characteristics and same genomic constitutions as durum wheat.

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Abbreviations: CIMMYT, International Maize and Wheat Improvement Center; HST, host selective toxin; QTL, quantitative trait locus; SHW, synthetic hexaploid wheat; SNB, Stagonospora nodorum blotch.

Tan spot and Stagonospora nodorum blotch (SNB), caused by the fungi *Pyrenophora tritici-repentis* (Died.) Drechs. and *Phaeosphaeria nodorum* (E. Müller) Hedjaroude [anamorph: *Stagonospora nodorum* (Berk.) Castellani & E. G. Germano], respectively, are two destructive foliar diseases of common wheat (*Triticum aestivum* L.) ($2n = 6x = 42$) and durum wheat (*T. turgidum* L. ssp. *durum*) ($2n = 4x = 28$). They both can cause yield losses as high as 50% during an epidemic (Riede et al., 1996; Fried and Meister, 1987). In recent years, tan spot and SNB have become quite common in many wheat production regions primarily because of climate changes and reduced tillage practices in many wheat growing regions of the world (Xu et al., 2004). Tan spot was identified as the most prevalent disease of wheat in Canada in 2003 (Tekauz et al., 2004), and Perello et al. (2003) indicated that tan spot has become more destructive in the southern Cone region of South America, including Argentina, Brazil, Chile, Paraguay, and Uruguay. Stagonospora nodorum blotch has been reported to occur in many parts of the world (Leath et al., 1993) and has become more common and important in some wheat production regions (DePauw, 1995).

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Growing resistant cultivars is considered the most effective strategy for controlling tan spot and SNB. Unfortunately, the majority of current durum and bread wheat cultivars are susceptible to both diseases due to their narrow genetic base (Lamari et al., 2005). Efforts to search for sources of resistance have been reported in a number of studies (Riede et al., 1996; Xu et al., 2004; Singh et al., 2006a,b; Wicki et al., 1999). Although complete resistances or immunity to the two diseases have not been identified, a high level of partial resistance to tan spot and SNB has been identified in synthetic hexaploid wheat (SHW) (Xu et al., 2004), bread wheat (Rees and Platz, 1990; Singh et al., 2006a,b), and its relative species such as *T. timopheevii* (Ma and Hughes, 1995), *T. monococcum* (Ma and Hughes, 1993), *Aegilops tauschii* (Ma and Hughes, 1993), *Ae. speltoides* (Ecker et al., 1990a), and *Ae. longissima* (Ecker et al., 1990b). However, high levels of resistance to both tan spot and SNB have not been identified in durum wheat germplasm. Xu et al. (2004) observed that almost all of the 35 durum wheat cultivars and breeding lines used as parents of International Maize and Wheat Improvement Center (CIMMYT) SHW lines are susceptible to the two diseases. In their recent studies, Singh et al. (2006a,b) evaluated a large number of wheat germplasm for resistance to tan spot and SNB and found no durum genotypes with a high level of resistance to both diseases.

Resistant sources of hexaploid wheat and wild relatives could be potentially used for durum wheat breeding. However, introgression of the resistance from hexaploid wheat to tetraploid durum wheat may not be always effective because the inheritance of complete resistance to tan spot and SNB in wheat, in most cases, is quantitative (Cao et al., 2001; Faris et al., 1997; Faris and Friesen, 2005; Friesen and Faris, 2004; Liu et al., 2004b). If gene interactions between the A or B genomes and the D genome contribute to some extent to resistance, or the major quantitative trait loci (QTLs) for resistance are located in D-genome chromosomes, introgression of the resistance could not be accomplished using conventional breeding approaches. Introgression of the resistance genes or major QTLs from alien species requires substantial efforts to induce homeologous recombination through chromosome engineering. Thus, the most useful source of tan spot and SNB resistance for durum wheat may be from other tetraploid wheat subspecies.

We recently evaluated 172 wild emmer accessions from Israel and identified 34 accessions with resistance to both tan spot and SNB (Chu et al., 2008), suggesting that other tetraploid wheat subspecies may possess resistance to the two diseases. This discovery motivated us to further evaluate the germplasm collections in five other tetraploid wheat subspecies, including *T. carthlicum*, *T. dicoccum*, *T. polonicum*, *T. turanicum*, and *T. turgidum*. Compared with wild emmer, these five subspecies are all in cultivated form, and their resistance, if identified, can be transferred into durum wheat using conventional breeding approaches.

The fungi causing tan spot and SNB both produce host selective toxins (HSTs). It has been demonstrated that HSTs are virulence factors and that disease severity usually correlates with sensitivity to the HSTs produced by the fungi. Host sensitivity to Ptr ToxA, a well-characterized toxin produced by *P. tritici-repentis*, has been found to be associated with disease susceptibility to *P. tritici-repentis* race 2 (Friesen et al., 2003; Lamari and Bernier, 1991). The dominant gene *Tsn1* controls sensitivity to Ptr ToxA, which is located on wheat chromosome arm 5BL (Faris et al., 1996). Genotypes without *Tsn1* are insensitive to the toxin (Anderson et al., 1999). By using partially purified SnTox1, a toxin predominantly produced by *P. nodorum*, Liu et al. (2004a,b) identified a gene, *Snn1*, conferring toxin sensitivity on chromosome arm 1BS, which explained as much as 58% of the phenotypic variation in SNB disease reaction. Friesen et al. (2006) indicated that the gene encoding Ptr ToxA in *P. tritici-repentis* was transferred from *P. nodorum* in a very recent horizontal gene transfer event. They noted a strong correlation between SnToxA sensitivity and SNB disease reaction. Therefore, the sensitivity of genotypes to major HSTs is an important factor in germplasm evaluation for resistance to both SNB and tan spot.

In this study, we attempted to identify new sources of tan spot and SNB resistance that can be easily used for durum wheat by evaluating a large number of accessions belonging to five cultivated tetraploid wheat subspecies, including *T. carthlicum*, *T. dicoccum*, *T. polonicum*, *T. turanicum*, and *T. turgidum* for reactions to *P. tritici-repentis* and *P. nodorum* and sensitivity to the HSTs produced by the two fungi.

MATERIALS AND METHODS

Plant Materials

A total of 688 accessions of cultivated tetraploid wheat were evaluated (Table 1). The collection consists of 97 *T. turgidum* L. subsp. *carthlicum* (Nevski) Á. Löve & D. Löve, 81 *T. turgidum* L. subsp. *polonicum* (L.) Thell, 200 *T. turgidum* L. subsp. *turgidum* (L.) Thell, 200 *T. turgidum* subsp. *dicoccum* (Shrank ex Schübler) Thell., and 110 *T. turgidum* L. subsp. *turanicum* (Jakubz.) Á. Löve & D. Löve accessions. The original seeds were kindly provided by Dr. Harold Bockelman USDA-ARS, National Small Grain Research Facility, National Small Grain Collection, Aberdeen, ID. In addition, the CIMMYT SHW line W-7976 and the hard red spring wheat cultivar Grandin were used as the resistant and susceptible checks, respectively.

Disease Screening Procedures

A randomized complete block design was used. All cultivated tetraploid accessions were first evaluated for reaction to *P. tritici-repentis* and *P. nodorum*, respectively, and then accessions with low or intermediate reaction types to either pathogen were selected and evaluated in two separate two-replicate experiments. Evaluation of reactions to *P. tritici-repentis* and *P. nodorum* were conducted under controlled greenhouse and growth chamber conditions using experimental procedures

Table 1. List of 688 tetraploid wheat (*Triticum turgidum* L. ssp.) accessions evaluated in this study.†

			***T. carthlicum* (97 accessions)**		
Cltr 7665	Cltr 7692	NSL70758–70761	PI 61102	PI 70738	PI 78812–78813
PI 94748–94755	PI 115816–115817	PI 168672	PI 182471	PI 190949	PI 251914
PI 272521–272522	PI 283887–283890	PI 286070–286071	PI 341800	PI 349040–349041	PI 352278–352282
PI 387696	PI 470729–470734	PI 499972	PI 532475–532502	PI 532504–532507	PI 532509–532518
PI 572848–572849	PI 573178–573182	PI 585017–585018			
			***T. polonicum* (81 accessions)**		
Cltr 13919	Cltr 14139–14140	Cltr 14803	Cltr 14869	Cltr 14892	Cltr 17442
PI 29447	PI 42209	PI 56261–56262	PI 134945	PI 167622	PI 185309
PI 190951	PI 191620	PI 191808	PI 191810	PI 191823	PI 191826
PI 191837	PI 191852	PI 191881	PI 191890	PI 191893	PI 191903
PI 192666	PI 208911	PI 210845	PI 223171	PI 225334–225335	PI 245663
PI 254214–254215	PI 266846	PI 272564–272570	PI 272572	PI 272590	PI 278647
PI 286547	PI 289606	PI 290512	PI 298572	PI 306548–306549	PI 330554–330555
PI 349051–349052	PI 352487–352489	PI 361757	PI 366117	PI 367198	PI 384265–384268
PI 384337–384345	PI 387457	PI 387479	PI 566593	PI 585015	PI 608017
PI 629119					
			***T. turgidum* (200 accessions)**		
Cltr 5988	Cltr 7688	Cltr 7772	Cltr 7774	Cltr 7778	Cltr 7785–7786
Cltr 7795–7796	Cltr 7798	Cltr 7809–7810	Cltr 7833	Cltr 7839–7841	Cltr 7859
Cltr 7863–7864	Cltr 7871	Cltr 7875	Cltr 7881	Cltr 7945	Cltr 8000
Cltr 8055	Cltr 8073	Cltr 8090	Cltr 8098–8099	Cltr 8107	Cltr 8109
Cltr 8115	Cltr 8155	Cltr 13712–13713	Cltr 14445	Cltr 14625	Cltr 14743
Cltr 14795	Cltr 14842	Cltr 14863	Cltr 17714	PI 28655	PI 32039
PI 41029	PI 52329	PI 56263	PI 57661–57662	PI 60617	PI 60715
PI 60729	PI 66058	PI 67339–67340	PI 94689	PI 125343	PI 134946–134949
PI 134951–134957	PI 134959–134962	PI 149812	PI 157983	PI 157985	PI 157986
PI 166484	PI 166496	PI 166591	PI 167502–167503	PI 167572	PI 167867
PI 173503	PI 178652	PI 185723–185724	PI 185726	PI 185728	PI 185734
PI 190928–190929	PI 190929	PI 190932	PI 190948	PI 190975	PI 190978–190980
PI 191015	PI 191104	PI 191145	PI 191203–191204	PI 191353–191354	PI 191389
PI 191445	PI 191534	PI 191579	PI 191871	PI 191885	PI 191904
PI 191951–191953	PI 191981	PI 192050	PI 192520	PI 208912	PI 210372
PI 210385	PI 211705	PI 212835	PI 213571	PI 220356	PI 221422–221425
PI 223173	PI 225308–225309	PI 234872	PI 245666	PI 245751	PI 255306
PI 264954	PI 264991	PI 264995	PI 265016	PI 266851	PI 266906
PI 272496	PI 272534	PI 272583–272584	PI 272587–272588	PI 272592–272593	PI 277125
PI 277127	PI 277679	PI 278221	PI 278367	PI 278596–278597	PI 278645
PI 286075	PI 290522	PI 290524	PI 290526–290528	PI 294568	PI 294574
PI 295011–295012	PI 295043	PI 295071–295074	PI 295351	PI 297859–297860	PI 297862
PI 306558–306564	PI 320139	PI 323440	PI 330560	PI 331258	PI 331262
PI 341283	PI 341300	PI 341332	PI 341391	PI 341482	PI 341608
PI 341611	PI 341615	PI 345413	PI 347131	PI 347133	
			***T. dicoccum* (200 accessions)**		
Cltr 3686	Cltr 4013	Cltr 7685–7687	Cltr 7779	Cltr 7962	Cltr 7966
Cltr 12213–12214	Cltr 14085–14086	Cltr 14098	Cltr 14133	Cltr 14135	Cltr 14137
Cltr 14437	Cltr 14454	Cltr 14592	Cltr 14621	Cltr 14636–14639	Cltr 14750–14752
Cltr 14787	Cltr 14822	Cltr 14824	Cltr 14834	Cltr 14838	Cltr 14866–14868
Cltr 14916–14917	Cltr 14919	Cltr 14970–14972	Cltr 17675–17676	NSL 70767	PI 2789
PI 11650	PI 40919	PI 41024–41025	PI 56234	PI 57394	PI 57536
PI 58788–58789	PI 60704–60706	PI 73388	PI 74104	PI 74106	PI 74108
PI 79899	PI 94613–94621	PI 94623–94628	PI 94630–94638	PI 94640–94642	PI 94648–94650
PI 94654–94671	PI 94673–94683	PI 94738	PI 94741	PI 94747	PI 101971
PI 113961	PI 113963	PI 133134	PI 154582	PI 164578	PI 164582
PI 168673–168679	PI 182743	PI 190920–190927	PI 190931	PI 191091	PI 191252
PI 191385–191387	PI 191390	PI 191781	PI 193641–193644	PI 193873	PI 193877–193883
PI 194041–194042	PI 194375	PI 195721–195723	PI 196099–196101	PI 196904–196905	PI 197259–197260
PI 197260	PI 197481–197496	PI 217637	PI 217639–217640	PI 221398–221400	

Table 1. Continued

T. turanicum (110 accessions)					
CItr 11390	CItr 14082	CItr 14089	CItr 14095	CItr 14598–14599	PI 10391
PI 67343	PI 68104	PI 68287	PI 68293	PI 113392–113393	PI 115814–115815
PI 124494	PI 125351	PI 127106	PI 166308	PI 166450	PI 166554
PI 166959	PI 167481	PI 182717	PI 184526	PI 184543	PI 185192–185193
PI 190973	PI 191599	PI 192641	PI 192658	PI 210383	PI 210386
PI 211668	PI 211691	PI 211708	PI 225328	PI 225330–225331	PI 251925
PI 254196–254199	PI 254201–254213	PI 256034	PI 257544	PI 272601–272602	PI 273985
PI 278350	PI 283795	PI 286069	PI 290530	PI 306665	PI 317491–317495
PI 321737	PI 321743	PI 330552	PI 337643	PI 341414	PI 347132
PI 349055	PI 352514–352515	PI 362067	PI 481582	PI 525355	PI 532136
PI 537992	PI 559976	PI 560896	PI 561075	PI 576854	PI 623629
PI 623641	PI 623656	PI 624207–624209	PI 624217	PI 624420–624422	PI 624429
PI 624892–624893	PI 625164	PI 625187	PI 625189	PI 625214	PI 625401
PI 627211					

†CItr and PI numbers indicate the accession number in USDA National Small Grains Collection, Aberdeen, ID.

described by Friesen et al. (2003) and Xu et al. (2004). A slight modification of the evaluation procedure is that in the first round of evaluation, both the resistant and susceptible checks were planted in three cones on the right border of each RL98 tray; the remaining cones around the border as well as cones inside were all planted with accessions to be tested.

The isolate Pti2 of *P. tritici-repentis* race 1 was used to produce inocula for evaluation of resistance to tan spot. *Pyrenophora tritici-repentis* race 1 is the most prevalent race in North America (Ali and Francl, 2003), and it also contains virulence factors found in race 2 (Lamari et al., 2003), the second most prevalent race (Ali and Francl, 2003). The isolate Pti2 was originally collected from a wheat field in South Dakota. Disease reactions were rated 7 d postinoculation using the 1 to 5 scale lesion-type rating system developed by Lamari and Bernier (1989), with 1 being resistant, 2 moderately resistant, 3 moderately resistant to moderately susceptible, 4 susceptible, and 5 highly susceptible. Lines showing equal number of two lesion types were given an intermediate reaction type (e.g., reaction type 1 and 2 equals 1.5).

For evaluation of reaction to *P. nodorum*, three diverse isolates, LDNSn4, BBCSn5, and Sn2000 were used to produce conidia. Three conidial suspensions were then equally mixed before inoculation. Sn2000 was collected from a North Dakota wheat field in 1980, and it has been shown to be an aggressive isolate that produces SnTox1 (Liu et al., 2004a) and SnToxA (Friesen et al., 2006). Sn2000 has been used to screen North Dakota wheat germplasm and breeding lines. Isolates LDNSn4 and BBCSn5, collected from North Dakota and Minnesota, respectively, produce other toxins in addition to those produced by Sn2000 (Friesen et al., 2007). Therefore, the mixture of these three isolates provides a variety of virulence factors present in *P. nodorum*. The concentration of conidial suspensions was adjusted to 1×10^6 conidia mL^{-1}, and plants were inoculated until runoff. The rating system used for *P. nodorum* is a qualitative numerical scale of 0 to 5 based on the lesion type as described in Liu et al. (2004b).

Toxin Infiltration

Toxin infiltration was done on those accessions with low disease reaction type after the first round of screening. At the two-leaf stage, plant leaves (three plants per line) were infiltrated with purified Ptr ToxA (provided by S.W. Meinhardt, Department of Plant Pathology, North Dakota State University, Fargo) and culture filtrate produced from Sn2000KO6-1, a strain generated from Sn2000 in which the SnToxA gene has been disrupted (Friesen et al., 2006). Sn2000 wild-type produces SnTox1 (Liu et al., 2004a) and SnToxA (Friesen et al., 2006), but with the disruption of *ToxA* gene, culture filtrates from Sn2000KO6–1 no longer contain SnToxA but still produce SnTox1 and potentially other unidentified host selective toxins. Toxin infiltration was done according to Xu et al. (2004). Leaves were evaluated 4 d after infiltration and scored as insensitive (–) or sensitive (+). Because SnToxA is functionally identical to Ptr ToxA (Friesen et al., 2006), the results from the Ptr ToxA infiltrations were considered the same as that for SnToxA infiltration.

Statistical Analysis

Statistical analysis was performed using the SAS version 9.1 (SAS Institute, 1999). Bartlett's χ^2 was calculated to test the homogeneity of variance in different replications. The least significant difference was used to test the significance of difference between the accessions as well as the checks. The two-sample *t* test was used to test the difference of average disease reactions to *P. tritici-repentis* and *P. nodorum* according to the reaction to HSTs. Regression analysis was performed to evaluate the correlation between sensitivity to HSTs and average reaction to *P. tritici-repentis* and *P. nodorum*. For regression analysis, the sensitivity was converted from sensitive and insensitive to 1 and 0, respectively.

RESULTS AND DISCUSSION

The evaluation data showed that different subspecies exhibited different reactions to *P. tritici-repentis* and *P. nodorum* (Table 2). Among the 97 *T. carthlicum* accessions, 42 had low or intermediate disease reactions to *P. nodorum* (≤2.5), but all were susceptible to tan spot. In contrast, 11 out of the 110 *T. turanicum* accessions had low or intermediate disease reactions to *P. tritici-repentis*, but no resistance to SNB was observed in this subspecies (Table 2).

Both *T. turgidum* and *T. dicoccum* appeared to be rich sources for resistance to both tan spot and SNB, particu-

larly in *T. dicoccum*, where 61 and 86 out of 200 accessions investigated showed low or intermediate disease reaction types (≤2.5) to *P. tritici-repentis* and *P. nodorum*, respectively. Furthermore, 35 accessions in *T. dicoccum* and 7 accessions in *T. turgidum* showed low to intermediate disease reactions (≤2.5) to both diseases. In total, 206 of 688 accessions had disease reaction types of less than 2.5 to either or both of the diseases in the first round of screening (Table 2). To verify the resistance, the 206 accessions were further evaluated for their reactions to *P. tritici-repentis*, *P. nodorum*, and HSTs produced by the two fungi in two additional experiments with two replications.

Table 2. Number of accessions with low or intermediate disease reaction (≤2.5) after the first round of screening in 688 tetraploid wheat (*Triticum turgidum* L. ssp) accessions investigated.

Species	No. of accessions	No. of accessions with disease reaction ≤ 2.5			
		Tan spot†	SNB‡	Tan spot and SNB	Total
T. carthlicum	97	0	42	0	42
T. polonicum	81	12	3	0	15
T. turgidum	200	10	23	7	26
T. dicoccum	200	61	86	35	112
T. turanicum	110	11	0	0	11
Total	688	94	154	42	206

†Caused by *Pyrenophora tritici-repentis* (Died.) Drechs.

‡Stagonospora nodorum blotch, caused by *Phaeosphaeria nodorum* (E. Müller) Hedjaroude [anamorph: *Stagonospora nodorum* (Berk.) Castellani & E. G. Germano].

Reaction to *Pyrenophora tritici-repentis* and Ptr ToxA in Accession Subsets

After the second round of evaluation of the subset of 206 accessions inoculated with *P. tritici-repentis* race 1 (Pti2) in two replicates, we then calculated Bartlett's χ^2 to test the variance homogeneity of disease reaction data from all three replications. Bartlett's χ^2 test indicated that the variance of disease reaction from all three replications was homogeneous ($\chi^2 = 3.58$, $P = 0.17$, df = 2); thus, the data were combined (Table 3). Twenty-five accessions had average disease reaction types less than 2 and are considered as resistant or partially resistant. The average disease reaction types of these accessions are shown in Table 4. Among these resistant accessions, 8, 13, and 4 were from *T. turgidum*, *T. dicoccum*, and *T. turanicum*, respectively. No resistance was identified among the *T. carthlicum* and *T. polonicum* accessions.

Results from Ptr ToxA infiltration in the subset show that 194 of the 206 accessions were insensitive, indicating that the majority of the accessions in the subset do not carry *Tsn1*. Since *Tsn1* has been found to be associated with increased disease susceptibility to both tan spot and SNB (Friesen et al., 2003, 2006; Lamari and Bernier, 1991), the presence of a limited number of accessions in the subset that carry *Tsn1* was likely due to the elimination of *Tsn1* genotypes from the first-round evaluation. The average disease reaction type of Ptr ToxA sensitive accessions was 3.2, which is slightly higher than that of insensitive accessions (3.0), suggesting that the sensitivity to Ptr ToxA can increase the susceptibility to *P. tritici-repentis*. But *t* test results showed that the difference was not significant (Table 5).

Sensitivity to Ptr ToxA has been reported to account for approximately 20% of the variation in tan spot disease reaction in a hexaploid wheat mapping population (Friesen et al., 2003). Xu et al. (2004) found the R^2 of 0.1 for association between sensitivity to Ptr ToxA and susceptibility to *P. tritici-repentis* in SHW lines. The results from our study suggest that *Tsn1* may have some positive effect on tan spot disease development in tetraploid wheat. The nonsignificant effect of sensitivity to Ptr ToxA on tan spot disease severity may have resulted from the genotype composites of the subset in which most of the accessions were insensitive to Ptr ToxA. Thus, the difference of tan spot reaction between Ptr ToxA sensitive and insensitive accessions may not truly reflect the effect of *Tsn1* on the development of tan spot. Alternatively, as indicated by Faris and Friesen (2005), Ptr ToxA may not be a significant factor in tan spot development. They reported race-nonspecific tan spot resistance QTLs on chromosomes 1B and 3B, but there was no effect of *Tsn1* on 5B. The results of this research indicate that genes other than *Tsn1* are involved with resistance to tan spot in many of the accessions evaluated, which is consistent with the result from *T. dicoccoides*, a wild relative of tetraploid wheat (Chu et al., 2008).

Reaction to *Phaeosphaeria nodorum*, SnToxA and Culture Filtrate in Subset Accessions

The Bartlett's χ^2 test showed that variance of disease reaction in the subset of 206 accessions to *P. nodorum* among the three replicates was homogeneous ($\chi^2 = 1.26$, $P = 0.53$, df = 2). Therefore, the disease reaction data from the three replicates were pooled and the number of accessions with average disease reactions ≤2 are shown in Table 4. A total of 132 out of 206 accessions showed resistance to SNB, with 59 accessions

Table 3. Number of resistant accessions with disease reaction type ≤2 in each tetraploid wheat subspecies (*Triticum turgidum* L. ssp.) when inoculating with *Pyrenophora tritici-repentis* and *Phaeosphaeria nodorum* at seedling stage after three replicates.

Species	No. of accessions resistant to		
	Tan spot†	SNB‡	Tan spot and SNB
T. carthlicum	0	37	0
T. polonicum	0	3	0
T. turgidum	8	21	5
T. dicoccum	13	71	5
T. turanicum	4	0	0
Total	25	132	10

†Caused by *Pyrenophora tritici-repentis* (Died.) Drechs.

‡Stagonospora nodorum blotch, caused by *Phaeosphaeria nodorum* (E. Müller) Hedjaroude [anamorph: *Stagonospora nodorum* (Berk.) Castellani & E. G. Germano].

Table 4. Average disease reaction to *Phaeosphaeria nodorum* (Sn) and *Pyrenophora tritici-repentis* (Ptr) and sensitivity to Ptr/SnToxA and culture filtrate (CF) of Sn2000KO6-1 in 147 tetraploid wheat (*Triticum turgidum* L. ssp) accessions that identified as resistant to either or both of the diseases from 206 subset accessions after three replicates.†

Accession no.‡	Line or cultivar name	Growth habit	Reaction to Sn§		Reaction to CF¶	Reaction to Ptr/Sn ToxA¶	Reaction to Ptr§	
			Avg.	SD			Avg.	SD
			***T. carthlicum* (37 accessions)**					
PI 94749	350	Spring	0.5	0.00	–	–	3.5	0.00
PI 94751	352	Spring	0.5	0.00	–	–	3.7	0.29
PI 286070		Spring	0.5	0.00	–	–	3.8	0.29
PI 532504	H83-1541-1	Spring	0.5	0.00	–	–	3.8	0.29
PI 352280	T-1514	Spring	0.5	0.00	–	–	4.8	0.29
PI 283887	Persian	Spring	0.7	0.29	–	–	3.0	0.00
PI 532502	H83-1579	Spring	0.7	0.29	+	–	3.0	0.00
PI 352278	T-1300	Spring	0.7	0.29	–	–	3.5	0.00
PI 349040	WIR 13810	Spring	0.7	0.29	–	–	4.5	0.00
PI 532507	H83-1534-6	Spring	0.8	0.29	–	–	3.3	0.29
PI 532487	79TK108–574, HD1	Spring	0.8	0.29	+	–	5.0	0.00
PI 283888	Persian	Spring	1.0	0.87	–	–	3.0	0.00
PI 532515	H84-561-1	Spring	1.0	0.87	–	–	4.0	0.00
PI 251914	WIR 25170	Spring	1.0	0.00	+	–	4.2	0.29
PI 341800	WIR 32510	Spring	1.0	0.50	–	–	4.3	0.29
PI 470729	79TK097-503	Spring	1.0	0.87	–	–	4.7	0.29
PI 532509	H83-1578	Spring	1.2	0.76	–	–	3.2	0.29
PI 352279	T-1513	Spring	1.2	0.76	+	–	3.5	0.00
PI 115816	7106	Spring	1.2	0.76	+	–	3.7	0.58
PI 286071		Spring	1.2	0.76	+	–	4.0	0.00
PI 61102	Rusak	Spring	1.3	0.76	–	–	3.0	0.00
PI 94753	354	Spring	1.3	0.76	+	–	3.5	0.50
PI 94752	353	Spring	1.3	0.76	–	–	4.2	0.29
PI 94748	349	Spring	1.3	0.29	+	–	4.3	0.29
PI 532492	79TK097-503, HD34	Spring	1.3	0.58	–	–	4.8	0.29
PI 532477	79TK098-517, HD1	Spring	1.3	0.29	+	–	5.0	0.00
PI 532501	H83-1537	Spring	1.5	0.87	+	–	3.3	0.29
PI 78812	Cltr 10110	Spring	1.5	0.50	–	–	4.0	0.00
PI 532495	79TK097-503, HD13	Spring	1.7	0.58	+	–	4.5	0.00
PI 532491	79TK103-544A-2	Spring	1.8	0.29	–	–	3.7	0.29
PI 532499	79TK100-532D-2	Spring	1.8	0.76	+	–	3.8	0.29
PI 532493	79TK098-517, HD5	Spring	1.8	0.76	+	–	4.3	0.29
PI 532488	79TK108-572-3, HD2	Spring	1.8	1.26	–	–	4.7	0.29
PI 352281	14-Sep9/14	Spring	2.0	0.00	+	–	4.0	0.00
PI 532505	H83-1538	Spring	2.0	0.50	+	–	4.0	0.00
PI 532482	79TK100-531A, HD2	Spring	2.0	0.50	–	–	4.8	0.29
PI 532481	79TK097-503, HD58	Spring	2.0	1.00	–	–	5.0	0.00
			***T. polonicum* (3 accessions)**					
PI 330555	Martinari	Spring	0.8	0.58	–	–	2.3	0.58
PI 286547	Husco	Spring	1.5	0.00	–	–	3.0	0.00
PI 349051	WIR 39297	Spring	2.0	0.50	+	–	3.0	0.00
			***T. turgidum* (24 accessions)**					
PI 210385		Winter	0.5	0.00	–	–	2.0	0.00
PI 134960	Pseudo-mirabile	Spring	0.5	0.00	–	–	3.0	0.00
PI 245666	k3158	Winter	0.5	0.00	–	–	3.2	0.29
PI 294574	Rampton Rivet	Winter	0.5	0.00	–	–	4.3	0.29
PI 341482	Sarki Karaagac	Winter	0.5	0.00	–	–	4.3	0.29
PI 191981	Bagudo	Spring	0.5	0.00	–	–	3.3	0.58
PI 191389	Macretherum	Winter	0.7	0.29	–	–	3.2	0.29
PI 290524	Rampton Rivet	Winter	0.7	0.29	–	–	4.2	0.29
PI 272588	I-1-3436		0.7	0.29	–	–	4.5	0.50
PI 306558	2956	Winter	0.7	0.29	–	–	4.7	0.29
PI 294568	Blue Cone	Winter	0.8	0.29	–	–	4.2	0.29
PI 190932	7.2481	Spring	1.0	0.00	+	–	1.7	0.29
PI 41029	533	Spring	1.0	0.50	–	–	3.8	0.29

Table 4. Continued.

Accession no.‡	Line or cultivar name	Growth habit	Reaction to Sn§		Reaction to CF¶	Reaction to Ptr/Sn ToxA¶	Reaction to Ptr§	
			Avg.	SD			Avg.	SD
PI 290528	Vermelho de Barba Preta	Spring	1.0	0.87	–	–	4.3	0.29
PI 190979	3786	Winter/Spring	1.2	0.29	–	–	1.8	0.29
PI 220356	Gandum	Spring	1.2	0.76	–	–	2.0	0.00
PI 134948	2800	Spring	1.2	0.29	–	–	3.3	0.29
PI 278221	Rivet	Winter	1.2	0.76	–	–	4.5	0.50
PI 134947	Gentile	Spring	1.3	0.29	–	–	3.5	0.50
Cltr 13712	Cole's Selection	Spring	1.5	0.50	–	–	2.0	0.00
PI 290522	Berkners Rauh	Winter	1.7	1.04	–	–	4.5	0.00
PI 190980	3878	Winter	2.7	0.29	–	–	1.7	0.29
PI 134961	Rubroatrum	Spring	2.8	0.29	–	–	1.8	0.29
PI 191445	2743	Spring	3.3	0.29	–	–	2.0	0.00
		***T. dicoccum* (79 accessions)**						
PI 94617	233	Spring	0.5	0.00	–	–	3.0	0.00
PI 41025	859	Spring	0.7	0.29	–	–	1.7	0.29
PI 74108	35900	Spring	0.7	0.29	–	–	2.2	0.29
PI 191252	Escana Doble Valverde de Jucar	Winter	0.7	0.29	–	–	2.3	0.29
PI 191781	Amylium	Spring	0.7	0.29	–	–	2.3	0.76
PI 197486	10179	Spring	0.7	0.29	–	–	3.0	0.00
PI 94626	243	Spring	0.7	0.29	–	–	4.8	0.29
PI 74104	35894	Spring	0.7	0.29	–	–	3.0	0.00
Cltr 14133		Spring	0.8	0.58	–	–	1.5	0.00
PI 94634	251	Spring	0.8	0.29	–	–	2.0	0.00
PI 94620	236	Spring	0.8	0.29	–	–	2.3	0.29
PI 56234	Cltr 7042	Spring	0.8	0.58	–	–	2.5	0.00
PI 57394	Cltr 7180	Spring	0.8	0.58	–	–	2.5	0.50
PI 94648	265	Spring	0.8	0.29	–	–	2.5	0.00
PI 94615	231	Spring	0.8	0.29	–	–	2.8	0.29
PI 58788	311	Spring	0.8	0.29	–	–	3.0	0.00
PI 190926	2475	Spring	0.8	0.29	–	–	3.2	0.29
PI 197493	10188	Spring	0.8	0.58	–	–	3.7	0.29
PI 182743	10399	Winter	1.0	0.50	+	–	1.3	0.29
PI 190922	2471	Spring	1.0	0.87	+	–	1.5	0.50
Cltr 14868	ELS 6404-142-3	Spring	1.0	0.50	+	–	2.2	0.29
PI 94642	259	Spring	1.0	0.50	–	–	2.5	0.00
PI 94657	274	Spring	1.0	0.00	–	–	2.5	0.00
PI 94659	276	Spring	1.0	0.50	–	–	2.7	0.29
Cltr 7685		Spring	1.0	0.50	–	–	2.8	0.29
Cltr 7686		Spring	1.0	0.50	–	–	3.0	0.00
PI 94680	372	Spring	1.0	0.00		–	3.0	0.00
Cltr 7966	493	Spring	1.0	0.50	–	–	3.0	0.00
PI 168675	16	Spring	1.0	0.00	+	–	3.0	0.00
PI 94660	277	Spring	1.2	0.29	–	–	2.2	0.76
Cltr 14834	ELS 6404-132	Spring	1.2	0.29	–	–	2.3	0.29
Cltr 14824	ELS 6404-129-2	Spring	1.2	0.76	+	–	2.7	0.29
PI 94667	293	Spring	1.2	0.76	–	–	2.8	0.29
PI 94673	298	Spring	1.2	0.76	+	–	3.0	0.00
PI 197489	10182	Spring	1.2	0.29	+	–	3.0	0.00
PI 190921	2467	Spring	1.2	0.29	–	–	3.2	0.29
PI 193644	8732	Spring	1.2	0.76	+	–	3.2	0.29
PI 94655	272	Spring	1.2	0.58	–	–	3.5	0.00
PI 197492	10185	Spring	1.2	0.58	–	–	3.7	0.29
Cltr 7962	493	Spring	1.2	0.29	–	–	3.5	0.50
PI 94675	302	Spring	1.3	0.76	–	–	2.2	0.29
PI 194042	8865	Spring	1.3	0.29	+	–	2.2	0.29
PI 94635	252	Spring	1.3	0.76	+	–	2.5	0.00
PI 191387	ST 1975	Spring	1.3	0.58	+	–	2.5	0.00
Cltr 14970		Spring	1.3	0.29	+	–	2.8	0.29

Table 4. Continued.

Accession no.‡	Line or cultivar name	Growth habit	Reaction to Sn§		Reaction to CF¶	Reaction to Ptr/Sn ToxA¶	Reaction to Ptr§	
			Avg.	SD			Avg.	SD
PI 191390	Rufum	Spring	1.3	0.58	–	–	2.8	0.29
CItr 14751	ELS 6404-108-5	Spring	1.3	0.58	+	–	3.0	0.00
PI 94618	234	Spring	1.3	0.76	–	–	3.0	0.00
PI 94640	257	Spring	1.3	0.29	–	–	3.0	0.00
CItr 14638	ELS 6404-78-2	Spring	1.3	0.29	+	–	3.3	0.29
PI 94632	249	Spring	1.3	0.29	+	–	3.7	0.29
PI 197485	10178	Spring	1.3	0.29	–	–	3.7	0.29
PI 197488	10181	Spring	1.3	0.29	–	–	3.7	0.29
PI 197484	10177	Spring	1.3	0.58	–	–	3.8	0.29
PI 94674	301	Spring	1.3	0.76	–	–	4.0	0.00
PI 94681	373	Spring	1.3	0.76	–	–	4.8	0.29
PI 168678	19	Spring	1.3	0.58	+	–	3.3	0.29
PI 197495	10190	Spring	1.5	0.50	+	–	2.8	0.29
PI 94614	230	Spring	1.5	0.50	–	–	3.0	0.00
CItr 14866	ELS 6404-142-1	Spring	1.5	0.50	+	–	3.3	0.29
PI 94671	297	Spring	1.5	0.50	+	–	3.3	0.29
PI 94633	250	Spring	1.5	0.50	–	–	3.8	0.29
PI 94662	279	Spring	1.5	0.50	–	–	3.8	0.29
CItr 3686	Vernal Emmer	Spring	1.7	0.29	–	–	3.0	0.00
CItr 14135	2669	Spring	1.7	0.58	–	–	3.0	0.00
PI 217640	13882	Spring	1.7	0.29	+	–	3.0	0.00
PI 94679	361	Spring	1.7	0.58	–	–	3.8	0.29
PI 193882	8933	Spring	1.8	0.29	+	–	3.0	0.00
PI 197491	10184	Spring	1.8	0.29	–	–	3.0	0.00
PI 193879	8930	Spring	1.8	0.29	+	–	3.2	0.29
PI 194041	8839	Spring	2.0	0.00	+	–	3.0	0.00
PI 190920	2323A	Spring	2.7	0.29	–	–	1.0	0.00
CItr 14137		Winter	3.0	0.00	–	–	1.5	0.00
CItr 17675	G 3081	Winter	3.0	0.00	–	–	1.5	0.50
CItr 14972		Spring	3.0	0.00	–	–	1.8	0.76
PI 193642	8568	Spring	3.0	0.00	+	–	2.0	0.00
PI 11650	Black Winter	Winter	3.0	0.00	+	–	1.7	0.29
PI 221398	Atratum	Winter	3.2	0.29	+	–	1.8	0.58
PI 94738	284	Spring	3.8	0.29	+	–	2.0	0.50
		***T. turanicum* (4 accessions)**						
PI 525355	1121	Spring	3.0	0.00	–	–	1.7	0.29
PI 341414	B-214	Spring	3.0	0.00	–	–	2.0	0.00
PI 362067	Jarcu I	Spring	3.2	0.29	+	–	2.0	0.00
PI 317495	Gondum Joharikoton	Spring	3.8	0.29	+	–	2.0	0.50
Checks								
	Grandin	Spring	4.3	0.29	+	+	4.5	0.00
	W7964	Spring	1.0	0.00	+	–	1.3	0.29
$LSD_{0.05}$			0.76				0.49	

†The accession number, line and cultivar name, and growth habits are specified based on USDA National Plant Germplasm System (http://www.ars-grin.gov/npgs/search-grin.html).

‡Accessions in italics are resistant to both diseases.

§Avg. = average disease reaction type of each accession in three replications.

¶Insensitive (–), sensitive (+); CF = culture filtrate from Sn2000KO6-1.

showing SNB disease reactions <1 (Table 4). Among these resistant accessions, 37 were *T. carthlicum*, 3 were *T. polonicum*, 21 were *T. turgidum*, and 71 were *T. dicoccum,* suggesting that these subspecies are good sources of SNB resistance. No resistance was identified from *T. turanicum*.

By viewing the reactions of the accessions to *P. tritici-repentis* and *P. nodorum*, we observed that five accessions each of *T. turgidum* (PI 210385, PI 190932, PI 190979, PI 220356, and CItr 13712) and *T. dicoccum* (PI 41025, PI 94634, PI 182743, PI 190922, and CItr 14133) showed average disease reactions <2 for both diseases. These would be especially useful for improving tan spot and SNB resistance simultaneously or for use as parental lines of mapping populations. The evaluation data also suggest that *T. dicoccum* has the largest number of

accessions resistant to both tan spot (13 accessions) and SNB (71 accessions). Recently, we also found that its wild relative, *T. dicoccoides*, is a rich source for tan spot and SNB resistance (Chu et al., 2008). Because *T. dicoccum* is most closely related to *T. dicoccoides*, some common tan spot and SNB resistance genes may possibly exist in both subspecies.

Reaction to toxins produced by *P. nodorum* was found to be correlated with the SNB disease reaction in the subset of 206 accessions investigated. The average SNB disease reactions of the accessions insensitive to SnToxA was 1.6, which was significantly (*t* test, $P < 0.0001$) lower than the 3.9 found in the sensitive accessions (Table 5). Simple linear regression analysis showed that SNB disease susceptibility was significantly associated with sensitivity to SnToxA (Table 6, $R^2 = 0.24$, $P < 0.0001$). The average disease reaction (1.4) of the accessions insensitive to Sn2000KO6-1 culture filtrate was also significantly (*t* test, $P < 0.0001$) lower than that (2.3) of sensitive accessions (Table 5). Simple linear regression analysis revealed that SNB disease reactions significantly correlate with sensitivity to the culture filtrate (Table 6, $R^2 = 0.12$, $P < 0.0001$).

The isolate Sn2000 can predominantly produce SnTox1 (Liu et al., 2004a) and SnToxA (Friesen et al., 2006). Isolate Sn2000KO6-1 was derived from Sn2000 by disrupting the SnToxA gene (Friesen et al., 2006). Thus, the reactions of the accessions to culture filtrate of Sn2000KO6-1 would mainly reflect sensitivity to SnTox1 but potentially other unidentified toxins present in the culture filtrates. Therefore, SNB disease reactions in the tetraploid wheat accessions investigated are strongly correlated with sensitivity to SnToxA and SnTox1 or additional toxins. We obtained similar results from evaluating *T. dicoccoides* accessions (Chu et al., 2008). These results are consistent with the reports of Friesen et al. (2006) and Liu et al. (2004b) in hexaploid wheat, indicating the genetic control of major resistance to SNB in tetraploid wheat may be the same as that found in hexaploid wheat; that is, SNB resistance in tetraploid wheat is mainly conferred by *Tsn1* on chromosome arm 5BL and *Snn1* on chromosome arm 1BS.

Multiple regression analysis on the sensitivity to both SnToxA and culture filtrate of Sn2000KO6-1 with SNB disease reaction revealed an increased association between sensitivity to the HSTs and susceptibility to SNB (Table 6, $R^2 = 0.30$, $P < 0.0001$), suggesting that the effects from *Tsn1* (SnToxA sensitive) and *Snn1* (SnTox1 sensitive) or other unidentified toxin sensitivity loci are additive. The difference of average disease reactions between the accessions insensitive to both SnToxA and Sn2000KO6-1 culture filtrate and the accessions insensitive to at least one of the HSTs is 1.0, and *t* test showed it to be significant at $P < 0.0001$ (Table 5). Thus, host insensitivity to both toxins could significantly increase its resistance to SNB, further indicating the additive effects of the SNB resistance from different genomic regions governing sensitivity to the toxins.

Table 5. Two sample *t* test to compare the difference of average reactions to *Pyrenophora tritici-repentis* and *Phaeosphaeria nodorum* based on the reactions to host selective toxins (HSTs) in 206 tetraploid wheat accessions investigated.

Comparison†	AVG1‡	AVG2‡	Difference§	*t* value	*P*
Reaction to *P. tritici-repentis*					
Ptr ToxA$^-$ vs. Ptr ToxA$^+$	3.0	3.2	0.2	1.01	0.31
Reaction to *P. nodorum*					
SnToxA$^-$ vs. SnToxA$^+$	1.6	3.9	2.3	13.32	<0.0001
CF$^-$ vs. CF$^+$	1.4	2.3	0.9	8.39	<0.0001
CF$^-$ SnToxA$^-$ vs. at least one toxin sensitive	1.4	2.4	1.0	9.92	<0.0001

†'–' and '+' indicate insensitive and sensitive to HSTs respectively; CF, culture filtrate from Sn2000KO6-1.

‡AVG1 and AVG2 are the average disease reactions of the first and second component in the comparison, respectively.

§Difference = AVG1 – AVG2.

Table 6. Regression analysis to evaluate the association of sensitivity to host selective toxins with average reaction to *Phaeosphaeria nodorum* in 206 cultivated tetraploid wheat accessions investigated.

Traits†	R^2	*P*
Reaction to *P. nodorum* SnToxA sensitivity	0.24	<0.0001
Reaction to *P. nodorum* CF sensitivity	0.12	<0.0001
Reaction to *P. nodorum* SnToxA and CF sensitivity	0.30	<0.0001

†CF, culture filtrate from Sn2000KO6-1.

In summary, 688 cultivated tetraploid wheat accessions belonging to *T. carthlicum*, *T. dicoccum*, *T. polonicum*, *T. turanicum*, and *T. turgidum* were evaluated for their seedling resistance to tan spot and SNB. A number of accessions with resistance to either of the diseases were identified, and 10 accessions showed resistance to both diseases. In addition, almost half of the accessions we investigated were also tested for their resistance to Fusarium head blight (caused by *Fusarium graminearum* Schwabe [teleomorph: *Gibberella zeae* (Schw.) Petch]), and a few accessions of *T. carthlicum* and *T. dicoccum* resistant to tan spot and/or SNB were also showed good resistance to Fusarium head blight (Oliver et al., 2008). Therefore, the resistant tetraploid wheat accessions investigated in this study may be useful for improving durum wheat for resistance to multiple fungal diseases. Since all the accessions are currently maintained in USDA National Small Grain Research Facility, evaluation data presented in this article can provide useful information for the selection of parental lines either for practical breeding or for developing mapping populations to identify the resistance genes and their associated molecular markers.

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